

AMENDMENTS TO THE CLAIMS

1. (Currently amended) A method for predicting the likelihood that a human ~~an individual~~ will have impaired or will not have impaired ~~enhanced~~ hippocampal function, assayed with functional magnetic resonance imaging (fMRI), comprising the steps of obtaining a DNA sample from a human ~~an individual~~ to be assessed and determining the presence or absence of a single nucleotide polymorphism from G to A resulting in the substitution of a methionine residue for a valine residue at amino acid position 66, relative to the start of the precursor protein sequence for brain-derived neurotrophic factor (BDNF), wherein a single nucleotide polymorphism from G to A resulting in the substitution of a methionine residue for a valine residue at amino acid position 66 (relative to the start of the precursor protein sequence) is indicative of the likelihood that a human will have ~~correlated with~~ impaired hippocampal function, assayed with fMRI, and a single nucleotide polymorphism from A to G resulting in the substitution of a valine residue for a methionine residue at amino acid position 66 (relative to the start of the precursor protein sequence) is indicative of the likelihood that a human will not have impaired ~~correlated with enhanced~~ hippocampal function, assayed with fMRI.

2. (Currently amended) A method for predicting the likelihood that a human ~~an individual~~ will have impaired or will not have impaired ~~enhanced~~ hippocampal dependent verbal memory, assayed with memory scores, comprising the steps of obtaining a DNA sample from a human ~~an individual~~ to be assessed and determining the presence or absence of a single nucleotide polymorphism from G to A resulting in the substitution of a methionine residue for a valine residue at amino acid position 66, relative to the start of the precursor protein sequence for brain-derived neurotrophic factor (BDNF), wherein a single nucleotide polymorphism from G to A resulting in the substitution of a methionine residue for a valine residue at amino acid position 66 (relative to the start of the precursor protein sequence) is indicative of the likelihood that a human will have ~~correlated with~~ impaired hippocampal dependent verbal memory, assayed with memory scores, and a single nucleotide polymorphism from A to G resulting in the substitution of a valine residue for a methionine residue at amino acid position 66 (relative to the start of the precursor protein sequence) is indicative of the likelihood that a human will not have impaired ~~correlated with enhanced~~ hippocampal dependent verbal memory, assayed with memory scores.

3. (Canceled)

4. (Original) A method according to Claim 1, wherein the human individual is a human an individual at risk for development of impaired hippocampal function.

5. (Original) The method according to Claim 2, wherein the human individual is a human an individual at risk for development of impaired hippocampal dependent verbal memory.

6. (Canceled)

7. (Original) A method according to Claim 1, wherein the human individual exhibits clinical symptomatology associated with impaired hippocampal function.

8. (Original) A method according to Claim 2, wherein the human individual exhibits clinical symptomatology associated with impaired hippocampal dependent verbal memory.

9. (Canceled)

10. (Original) A method according to Claim 1, wherein the human individual has been clinically diagnosed as having impaired hippocampal function.

11. (Original) A method according to Claim 2, wherein the human individual has been clinically diagnosed as having impaired hippocampal dependent verbal memory.

12. (Canceled)

13. (Currently amended) A method for predicting the likelihood than a human an individual will have impaired or will not have impaired enhanced hippocampal function, assayed with functional magnetic resonance imaging (fMRI), comprising the steps of obtaining a biological sample from a human an individual to be assessed containing the precursor BDNF protein or relevant portion thereof and determining the amino acid present at amino acid position +66 relative to the first amino acid of the precursor protein, wherein the presence of methionine at this position is indicative of the likelihood that a human will have impaired hippocampal function, assayed with fMRI, and the presence of valine at this position is indicative of the likelihood that a human will not have impaired enhanced hippocampal function, assayed with fMRI.

14. (Currently amended) A method for predicting the likelihood that a human an individual will have impaired or will not have impaired enhanced hippocampal dependent verbal memory, assayed with memory scores, comprising the steps of obtaining a biological sample from a human an individual to be assessed containing the precursor BDNF protein or relevant portion thereof and determining the amino acid present at amino acid position +66 relative to the

first amino acid of the precursor protein, wherein the presence of methionine at this position is indicative of the likelihood that a human will have impaired hippocampal dependent verbal memory, assayed with memory scores, and the presence of valine at this position is indicative of the likelihood that a human will not have impaired ~~enhanced~~ hippocampal dependent verbal memory, assayed with memory scores.

15. (Canceled)

16. (Original) A method according to Claim 13, wherein the human individual is a human ~~an individual~~ at risk for development of impaired hippocampal function.

17. (Original) The method according to Claim 14, wherein the human individual is a human ~~an individual~~ at risk for development of impaired hippocampal dependent verbal memory.

18. (Canceled)

19. (Original) A method according to Claim 13, wherein the human individual exhibits clinical symptomatology associated with impaired hippocampal function.

20. (Original) A method according to Claim 14, wherein the human individual exhibits clinical symptomatology associated with impaired hippocampal dependent verbal memory.

21. (Canceled)

22. (Original) A method according to Claim 13, wherein the human individual has been clinically diagnosed as having impaired hippocampal function.

23. (Original) A method according to Claim 14, wherein the human individual has been clinically diagnosed as having impaired hippocampal dependent verbal memory.

24. (Canceled)

25. (Withdrawn) A method for screening compounds useful for modulation of hippocampal function, comprising the steps of contacting a compound with a cultured host cell or membrane thereof that expresses a BDNF receptor or domain thereof and detecting binding of said compound to the BDNF receptor for domain thereof, whereby said binding identifies said compound as a candidate useful for modulation of hippocampal function.

26. (Withdrawn) The method of claim 25, which further comprises conducting the identification of the compound in the presence of labeled or unlabeled BDNF or homolog thereof.

27. (Withdrawn) A method for screening compounds useful for modulation of verbal memory, comprising the steps of contacting a compound with a cultured host cell or membrane thereof that expresses a BDNF receptor or domain thereof and detecting binding of said compound to the BDNF receptor for domain thereof, whereby said binding identifies said compound as a candidate useful for modulation of verbal memory.

28. (Withdrawn) The method of claim 27, which further comprises conducting the identification of the compound in the presence of labeled or unlabeled BDNF or homolog thereof.

29. (Withdrawn) A method for screening compounds useful for modulation of risk for schizophrenia, schizoaffective disorder, and other psychotic and mental disorders involving impaired memory, comprising the steps of contacting a compound with a cultured host cell or membrane thereof that expresses a BDNF receptor or domain thereof and detecting binding of said compound to the BDNF receptor for domain thereof, whereby said binding identifies said compound as a candidate useful for modulation of risk for schizophrenia, schizoaffective disorder, and other psychotic and mental disorders involving impaired memory.

30. (Withdrawn) The method of claim 29, which further comprises conducting the identification of the compound in the presence of labeled or unlabeled BDNF or homolog thereof.

31. (Withdrawn) A method for screening compounds useful for modulation of hippocampal function, comprising the steps of contacting a compound with a cultured host cell or membrane thereof that expresses a BDNF receptor or domain thereof, in the presence of labeled or unlabeled BDNF or homolog thereof, and determining whether said compound changes binding of said BDNF or homolog thereof to said BDNF receptor or domain thereof by measuring an amount of said BDNF or homolog thereof bound to said BDNF receptor or domain thereof, and identifying said compound as a candidate useful for modulation of hippocampal function, whereby said compound causes a change in binding of said BDNF or homolog thereof.

32. (Withdrawn) A method for screening compounds useful for modulation of verbal memory, comprising the steps of contacting a compound with a cultured host cell or membrane thereof that expresses a BDNF receptor or domain thereof, in the presence of labeled or unlabeled BDNF or homolog thereof, and determining whether said compound changes binding

of said BDNF or homolog thereof to said BDNF receptor or domain thereof by measuring an amount of said BDNF or homolog thereof bound to said BDNF receptor or domain thereof, and identifying said compound as a candidate useful for modulation of verbal memory, whereby said compound causes a change in binding of said BDNF or homolog thereof.

33. (Withdrawn) A method for screening compounds useful for modulation of risk for schizophrenia, schizoaffective disorder, and other psychotic and mental disorders involving impaired memory, comprising the steps of contacting a compound with a cultured host cell or membrane thereof that expresses a BDNF receptor or domain thereof, in the presence of labeled or unlabeled BDNF or homolog thereof, and determining whether said compound changes binding of said BDNF or homolog thereof to said BDNF receptor or domain thereof by measuring an amount of said BDNF or homolog thereof bound to said BDNF receptor or domain thereof, and identifying said compound as a candidate useful for modulation of risk for schizophrenia, schizoaffective disorder, and other psychotic and mental disorders involving impaired memory, whereby said compound causes a change in binding of said BDNF or homolog thereof.

34. (Withdrawn) A method for screening compounds useful for modulation of hippocampal function, comprising the steps of contacting a compound with a transgenic animal that expresses an exogenous BDNF gene and/or has one or both alleles of an endogenous BDNF gene inactivated, and detecting a change in said transgenic animal, whereby said change identifies said compound as a candidate useful for modulation of hippocampal function.

35. (Withdrawn) The method of claim 34, wherein the transgenic animal is a transgenic mouse.

36. (Withdrawn) A method for screening compounds useful for modulation of verbal memory, comprising the steps of contacting a compound with a transgenic animal that expresses an exogenous BDNF gene and/or has one or both alleles of an endogenous BDNF gene inactivated, and detecting a change in said transgenic animal, whereby said change identifies said compound as a candidate useful for modulation of verbal memory.

37. (Withdrawn) The method of claim 36, wherein the transgenic animal is a transgenic mouse.

38. (Withdrawn) A method for screening compounds useful for modulation of risk for schizophrenia, schizoaffective disorder, and other psychotic and mental disorders involving impaired memory, comprising the steps of contacting a compound with a transgenic animal that expresses an exogenous BDNF gene and/or has one or both alleles of an endogenous BDNF gene inactivated, and detecting a change in said transgenic animal, whereby said change identifies said compound as a candidate useful for modulation of risk for schizophrenia, schizoaffective disorder, and other psychotic and mental disorders involving impaired memory.

39. (Withdrawn) The method of claim 38, wherein the transgenic animal is a transgenic mouse.

40. (Withdrawn) A method of making a pharmaceutical composition useful for modulation of hippocampal function, comprising combining a pharmaceutically acceptable excipient and a compound identified by any of the preceding screening methods of Claims 25, 26, 31, 34, or 35.

41. (Withdrawn) A method of making a pharmaceutical composition useful for modulation of verbal memory, comprising combining a pharmaceutically acceptable excipient and a compound identified by any of the preceding screening methods of Claims 27, 28, 32, 36, or 37.

42. (Withdrawn) A method of making a pharmaceutical composition useful for modulation of risk for schizophrenia, schizoaffective disorder, and other psychotic and mental disorders involving impaired memory, comprising combining a pharmaceutically acceptable excipient and a compound identified by any of the preceding screening methods of Claims 29, 30, 33, 38, or 39.

43. (Withdrawn) A method of modulating hippocampal function in an individual, comprising administering a compound to the individual in an amount sufficient to mimic or inhibit binding of a BDNF receptor by endogenous BDNF, whereby hippocampal function is modulated.

44. (Withdrawn) A method of modulating verbal memory in an individual, comprising administering a compound to the individual in an amount sufficient to mimic or inhibit binding of a BDNF receptor by endogenous BDNF, whereby verbal memory is modulated.

45. (Withdrawn) A method of modulating risk for schizophrenia, schizoaffective disorder, and other psychotic and mental disorders involving impaired memory in an individual, comprising administering a compound to the individual in an amount sufficient to mimic or inhibit binding of a BDNF receptor by endogenous BDNF, whereby risk for schizophrenia, schizoaffective disorder, and other psychotic and mental disorders involving impaired memory is modulated.

46. (Withdrawn) A method of modulating hippocampal function in an individual, comprising administering a compound identified by any of the preceding screening methods of Claims 25, 26, 31, 34, or 35 to the individual in an amount sufficient to modulate hippocampal function.

47. (Withdrawn) A method of modulating verbal memory in an individual, comprising administering a compound identified by any of the preceding screening methods of Claims 27, 28, 32, 36, or 37 to the individual in an amount sufficient to modulate verbal memory.

48. (Withdrawn) A method of modulating risk for schizophrenia, schizoaffective disorder, and other psychotic and mental disorders involving impaired memory in an individual, comprising administering a compound identified by any of the preceding screening methods of Claims 29, 30, 33, 38, or 39 to the individual in an amount sufficient to modulate risk for schizophrenia, schizoaffective disorder, and other psychotic and mental disorders involving impaired memory.

49. (Withdrawn) A pharmaceutical composition, comprising a compound identified by any of the preceding screening methods of Claims 25, 26, 31, 34, or 35 in combination with a pharmaceutically acceptable excipient.

50. (Withdrawn) A pharmaceutical composition, comprising a compound identified by any of the preceding screening methods of Claims 27, 28, 32, 36, or 37 in combination with a pharmaceutically acceptable excipient.

51. (Withdrawn) A pharmaceutical composition, comprising a compound identified by any of the preceding screening methods of Claims 29, 30, 33, 38, or 39 in combination with a pharmaceutically acceptable excipient.